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Imaging in Acute Myocardial Infarction

The Pathologic Basis of Q-Wave and Non–Q-Wave Myocardial Infarction

A Cardiovascular Magnetic Resonance Study

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OBJECTIVES The purpose of this study was to determine the pathologic basis of Q-wave (QW) and non–Q-wave (NQW) myocardial infarction (MI).

BACKGROUND The QW/NQW distinction remains in wide clinical use but the meaning of the difference remains controversial. We hypothesized that measurement of total MI size and transmural extent by late gadolinium enhancement cardiovascular magnetic resonance (CMR) would identify the pathologic basis of QWs.

METHODS A total of 100 consecutive patients with documented previous MI had electrocardiogram and CMR on the same day. Patients with acute MI within seven days were excluded. Left ventricular function and the size and transmural extent of MI were quantified in the three major arterial territories and correlated with the presence of QW.

RESULTS Subendocardial MI showed QW in 28%. Transmural MI showed NQW in 29%. Of all MIs, 48% were at some point transmural, and 99% of these were at some point non-transmural. As MI size and number of transmural segments increased, the probability of QW increased (anterior: total size chi-square = 53, $p < 0.0001$, transmural extent chi-square = 36, $p < 0.0001$; inferior: total size chi-square = 16, $p = 0.001$, transmural extent chi-square = 10, $p = 0.001$). These findings did not hold for lateral MI. In a multivariate model, the transmural extent of MI was not an independent predictor of QW when total size of MI was removed. The QW/NQW classification was a good test for size of MI (area under receiver operating characteristic curve: anterior 0.90, inferior 0.77).

CONCLUSIONS The QW/NQW distinction is useful, but it is determined by the total size rather than transmural extent of underlying MI. (J Am Coll Cardiol 2004;44:554–60) © 2004 by the American College of Cardiology Foundation

For approximately 50 years, it has been clinical practice to stratify patients into Q-wave (QW) and non–Q-wave (NQW) myocardial infarction (MI) based on the electrocardiogram (ECG), but this remains controversial (1). In 1954, Prinzmetal reported that in an animal model, QW MIs were transmural and NQW MIs were subendocardial (2). Subsequent work questioned this distinction (3), and human autopsy study suggested that the association between QWs and the transmural extent of MI was random (4) and indeed a myth (5). Others suggested that the QW/NQW division was meaningless (5–7), although many anatomic and clinical studies showed that QW MIs were larger (8–10). The confusion is confounded by the presence of a variety of different published criteria for QW/NQW MI (11,12), which are at times misquoted (12,13) and a similar variety of definitions of transmural infarction (14).

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In current clinical practice, acute MI is divided into ST-segment and non–ST-segment elevation myocardial infarction (12). ST-segment and non–ST-segment elevation myocardial infarction ultimately develop with little crossover into QW and NQW MI, respectively (15). For ECG diagnosis of old infarction, the presence of QWs remains widely used clinically in guidelines (16–18) and in research, despite the lack of agreement on either the definition or anatomic basis of the distinction.

Recently a new technique for imaging of MI in vivo has been developed that might resolve this controversy. Late gadolinium enhancement cardiovascular magnetic resonance (CMR) allows the precise in vivo detection of the total size, location, and transmural extent of MI (19). The presence of myocardial enhancement indicates dead myocardium, and the technique has been validated against animal models of both

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Abbreviations and Acronyms

ACC	= American College of Cardiology
CMR	= cardiovascular magnetic resonance
ECG	= electrocardiogram
ESC	= European Society of Cardiology
MI	= myocardial infarction
QW/NQW	= Q-wave/non–Q-wave
ROC	= receiver operating characteristic
TIMI	= Thrombolysis In Myocardial Infarction

acute and chronic MI (20,21). We hypothesized that late gadolinium enhancement CMR would clarify the in vivo relationship between QW/NQW MI classification and both total size and transmural extent of MI.

METHODS

Patients. A total of 100 consecutive patients with previous MI undergoing CMR for clinical or research indications were enrolled in a prospective study. The exclusion criteria were the presence of any cardiac disease that may cause QW (e.g., hypertrophic cardiomyopathy, Wolff-Parkinson-White syndrome, dilated cardiomyopathy), and contraindications to CMR. Nine patients were not analyzed because of the presence of left bundle-branch block that prevents the interpretation of QW. No patient was excluded for poor image quality or technical limitations. No patient was scanned within seven days of acute MI when QW may be dynamic (22,23). A transmural MI was diagnosed when transmural enhancement was present at any point in the territory. The patient baseline characteristics are shown in Table 1.

CMR. The CMR was performed as previously described (24), using a 1.5-T scanner (Siemens Sonata, Erlangen, Germany) utilizing front and back surface coils and prospective ECG triggering. Briefly, after cine imaging in two long-axis views and up to 10 short-axis slices covering the entire myocardium, an intravenous bolus of 0.1 mmol/kg gadolinium-diethylenetriaminepentaacetic acid (Schering, Berlin, Germany) was given, and late enhancement images were acquired after 10 min in the same views as those used for cine imaging using an inversion-recovery sequence (25). Typical voxel size was $1.7 \times 1.4 \times 8$ mm. A complete series of short-axis late enhancement images is shown in Figure 1.

Table 1. Patient Baseline Characteristics

	Number (%)
Male	80 (88%)
Age (range)	62 ± 10 (33–83)
MI type*	
Recent (day 7–30)	33 (36%)
Chronic (>day 30)	41 (51%)
Known multiple	17 (13%)
Main location of MI†	
Anterior	43 (47%)
Inferior	18 (20%)
Lateral	18 (20%)
Multiple	12 (13%)
Ventricular function	
EDV (ml)	162 ± 68 (76–414)
ESV (ml)	88 ± 63 (15–338)
EF (%)	50 ± 16 (16–83)
Mass (g)	171 ± 48 (83–306)

Data are presented as n (%) or mean ± SD (range).
*From the patient history. †From cardiovascular magnetic resonance scans.
EDV = end-diastolic volume; EF = ejection fraction; ESV = end-systolic volume; MI = myocardial infarction.

Image analysis. Left ventricular function, volumes, and mass were calculated from the cine images using a three-dimensional analysis package (CMRTools, Cardiovascular Imaging Solutions, London, United Kingdom). For infarct assessment, the standard left ventricular 17-segment model was used (26). For each segment, the total amount of enhancement within each segment, irrespective of distribution, was scored on a six-point scale; 0: no hyperenhancement; 1: 0% to 25% infarction; 2: 26% to 50% infarction; 3: 51% to 75% infarction; 4: 76% to 99% infarction; 5: 100% infarction. Each segment was also assessed for the presence at any point of transmural MI. Scoring was performed by two observers. We validated this technique in 20 MI comparing semiquantitative scoring with planimetry and showed good correlation for infarct sizing ($R = 0.97$). Segments were grouped into three territories (anterior, inferior, and lateral) corresponding to the territories derived from ECG analysis and broadly to the perfusion zones of the left anterior descending, right coronary artery, and left circumflex (26). The anterior territory consists of seven segments, the inferior territory of five segments, and the lateral territory consists of five segments (Fig. 2). The total size of MI was calculated for each territory and expressed in quintiles.

ECG analysis. The ECG analysis was performed blindly without the knowledge of the CMR. Measurement for defining QW was undertaken according to the Minnesota Code (27). All measurements were performed using a loupe, and QW were adjudicated by consensus of two investigators. We used the QW/NQW MI definition from the Thrombolysis In Myocardial Infarction (TIMI) group in the primary analysis which defines QW MI as pathologic QW (>30 ms) in ≥2 contiguous leads (11). Three MI territories were considered: Anterior: I, aVL, V_1 to V_6. Inferior: II, III, aVF. Lateral MI was not defined by the TIMI group, so we used a definition of QW MI equivalent of a predominant R in V_1 and/or V_2 with an R ≥0.04 ms and no right axis deviation ≥100° or right bundle-branch block to avoid false positives (28–30).

Because there is more than one definition of anterior and inferior QW MI in clinical use, we also compared the TIMI definition with the European Society of Cardiology/American College of Cardiology (ESC/ACC) redefined consensus definition of QW MI (12,13). The recommendations were interpreted as follows: an anterior QW MI was either any QW (regardless of duration and depth) in all leads V_1 through V_3 or two contiguous QWs ≥30 ms in duration and ≥1 mm in depth in leads I, aVL, V_4, V_5, or

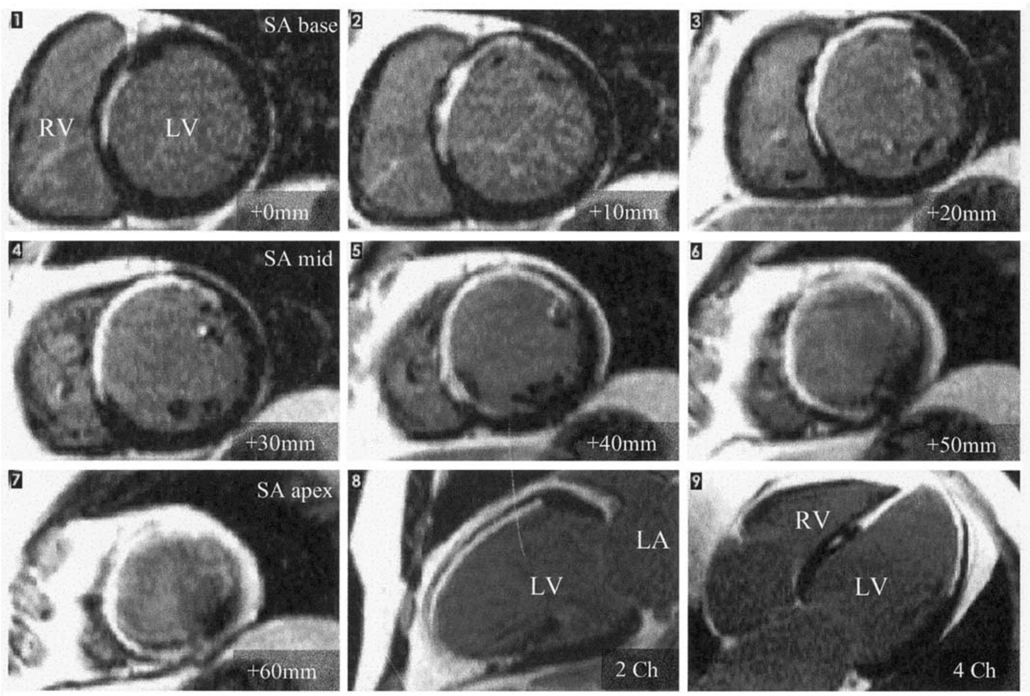

Figure 1. A representative complete late gadolinium enhancement study. This patient had a first anterior myocardial infarction (MI) four months previously owing to an occluded left anterior descending coronary artery. The MI was quantified as 42% of the total myocardium (71% of anterior territory, 36% of inferior, and 8% of lateral). The MI was transmural in four of the seven segments in the anterior territory. LA = left atrium; LV = left ventricle; RV = right ventricle; SA = short axis.

V_6; an inferior QW MI was defined as requiring QWs ≥30 ms duration and ≥1 mm in depth in both II and aVF (lead III is not used).

Statistical analysis. Two-tailed *t* tests were used to compare continuous variables, which are expressed as means ± SD. The chi-square test for trend was used to examine the influence of the transmural extent and the size of MI on the presence of QWs. Receiver operating characteristic (ROC) curves were used to assess the relationship between classification as QW/NQW MI and the total size or the transmural extent of MI. Areas under the ROC curves were compared using the technique described by DeLong et al. (31). Multivariate analysis included the number of transmural segments and size of MI as independent variables, and outcome classification as QW or NQW MI.

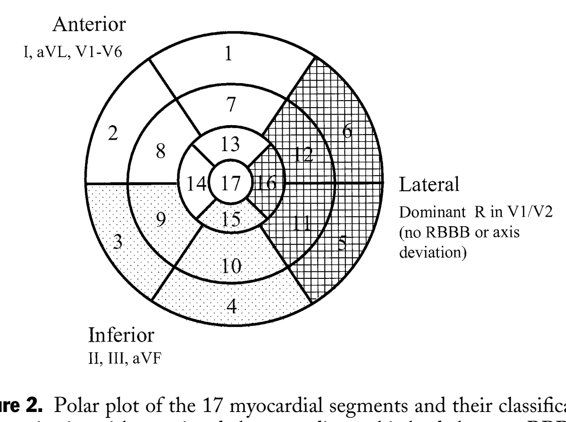

Figure 2. Polar plot of the 17 myocardial segments and their classification into territories with associated electrocardiographic lead changes. RBBB = right bundle-branch block.

RESULTS

QWs and total size of MI. As the total size of MI increased in the anterior and inferior territories (but not lateral), the probability of classification as QW MI increased (anterior, chi-square = 53, $p < 0.0001$; inferior, chi-square = 16, $p = 0.001$; lateral, chi-square = 2.2, $p = 0.69$) (Fig. 3). Classification into QW/NQW MI was a good diagnostic test for size of MI; the area under an ROC curve was 0.90 for the anterior territory and 0.77 for the inferior territory (Fig. 4). There was no relationship in the lateral territory. The area under an ROC curve was 0.85 for all MIs. The QW MIs were larger than NQW MIs (anterior: 54% vs. 29% of territory infarcted, $p < 0.0001$; inferior: 34% vs. 22%, $p = 0.007$).

QWs and transmural extent of MI. As the extent of transmural MI increased, the probability of classification as QW MI increased. (anterior, chi-square = 36, $p < 0.0001$; inferior, chi-square = 10, $p = 0.001$) (Fig. 3). The area under ROC curves was 0.83 for the anterior territory and 0.70 for the inferior territory. There was no relationship in the lateral territory. The area under an ROC curve was 0.79 for all MIs.

Total size versus transmural extent and QWs. The total size of MI was a statistically better predictor of QW/NQW MI classification than transmural extent (anterior: area under ROC curve: 0.90 vs. 0.83, $p = 0.03$; inferior: 0.77 vs. 0.70, $p = 0.02$). Multivariate analysis demonstrated that the transmural extent did not significantly increase the area under ROC curves when added to the total size of MI

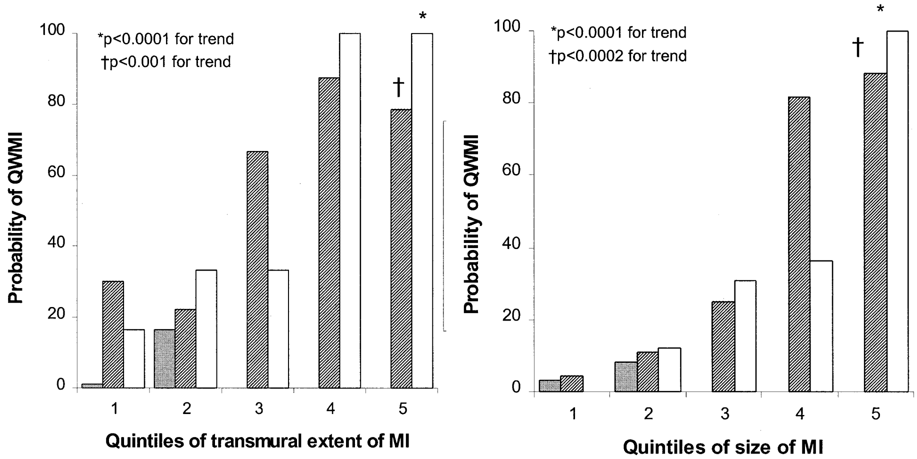

Figure 3. The probability of classification as Q-wave (QW) myocardial infarction (MI) by Thrombolysis In Myocardial Infarction (TIMI) criteria plotted against the quintiles of transmural extent **(left graph),** or total MI size **(right graph).** As both the transmural extent and the total size of MI increases, so the likelihood of classification as QW MI increases, for anterior and inferior but not lateral territories. **Gray bars** = lateral; **cross-hatched bars** = anterior; **white bars** = inferior.

(anterior, p = 0.73; inferior, p = 0.30) and was not an independent predictor of classification into QW/NQW MI in either territory.

QWs and function. Patients classified as having had any previous QW MI had a lower ejection fraction than anterior NQW MI (47% vs. 55%, p = 0.02). Patients with anterior QW MI had lower ejection fractions (45% vs. 55%, p = 0.003), but there was no difference for inferior QW MI (49% vs. 51%, p = NS).

Comparison of TIMI and ESC/ACC definitions of QW/NQW MI. The ESC/ACC definition of QW/NQW MI resulted in fewer patients classified as QW MI (anterior: 40 patients vs. 26; inferior: 18 patients vs. 13). Despite this, ROC analysis demonstrated that both the TIMI and the ESC/ACC definitions correlate with MI size

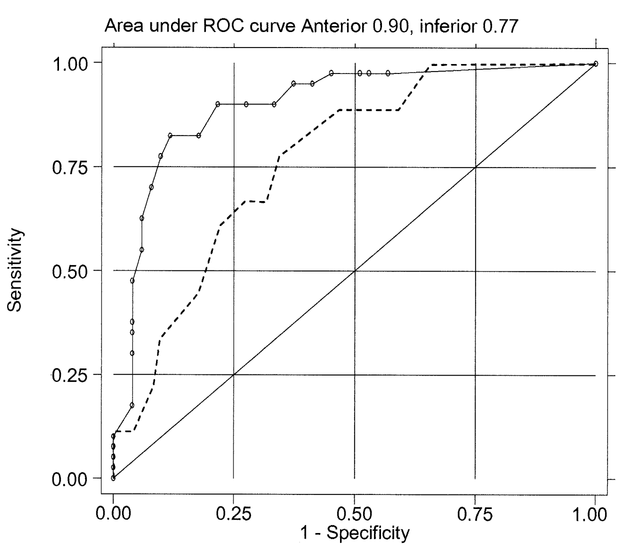

Figure 4. Receiver operating characteristic (ROC) curve analysis of infarct size as a predictor of the presence of Q waves (Thrombolysis In Myocardial Infarction [TIMI] criteria) for anterior **(solid line)** and inferior **(dashed line)** territories.

(anterior: TIMI 0.90 vs. ESC/ACC 0.87, p = 0.58; inferior TIMI 0.77 vs. ESC/ACC 0.75, p = 0.83).

Lateral infarction and other ECG changes. The size of lateral infarction and the presence of QW in I, aVL, V_5, and V_6 were also tested independently and together against the size of lateral MI. No significant relationships were found.

Do subendocardial MIs have QWs? Of anterior MIs, 6 of 21 (28%) with no transmural region and 34 of 48 (70%) with a transmural region were classified QW MI (Fig. 5). Therefore, equating NQW MI with subendocardial MI and QW MI with transmural MI would be wrong for approximately one-third of subendocardial MIs and a third of transmural anterior MIs. Of all MI territories, 48% were at some point transmural, and 96 of 97 (99%) of MIs with a transmural segment also had at least one non-transmural segment, making classification into subendocardial/ transmural overly simplistic (Fig. 5).

DISCUSSION

There are two main results of this study: first, that the QW/NQW distinction is useful clinically; and second, that the primary determinant of the presence of a QW is the total size of the underlying territorial infarction rather than its transmural extent. The larger the MI, the more likely there is to be QW on the ECG. Conversely, finding a QW MI by ECG is a good test for a large MI. The transmural extent of MI also correlates to the presence of QW MI, but transmural MI is neither a necessary condition for QW nor independent of total MI size. Additionally, the data emphasize that MIs have a complex structure with a varying transmural extent, making a transmural/non-transmural division over-simplistic. The electrical silence of the lateral territories to a standard 12-lead ECG is not surprising given the anterior chest wall lead placement, and fits with evidence that lateral infarction may present with little ECG alteration (32).

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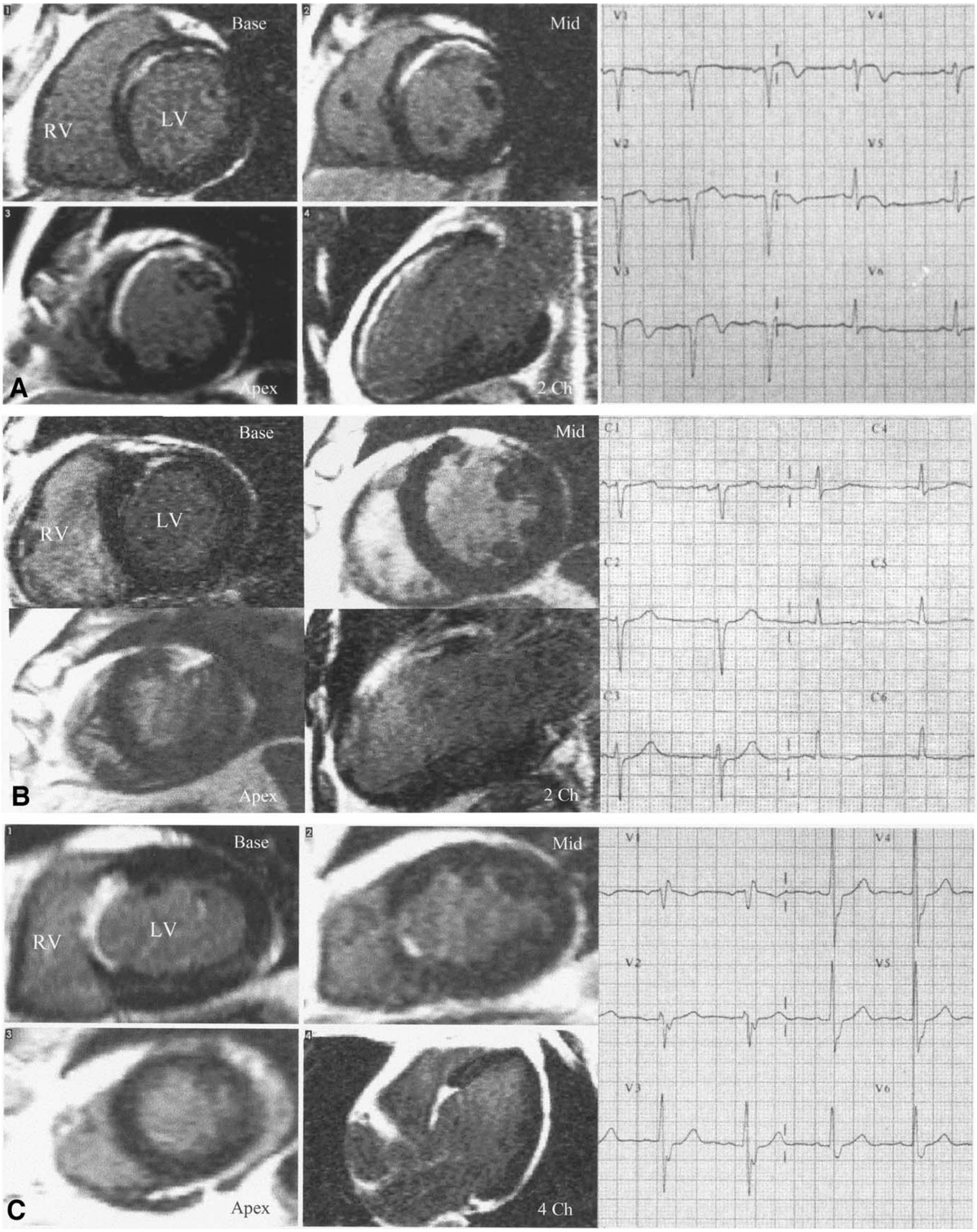

Figure 5. Three anterior MIs with contrast images and electrocardiographic leads V_1 to V_6 shown: **(A)** non-transmural Q-wave (QW) myocardial infarction (MI), **(B)** transmural non–Q-wave (NQW) MI, **(C)** transmural NQW MI with right bundle-branch block. These cases illustrate the relative importance of the total size of infarction over the transmural extent of infarction. The non-transmural MI shows QWs, with 24% of the left ventricle (LV) infarcted, whereas the transmural MIs do not have QWs, with 9% and 13% of the LV infarcted, respectively. The infarct localized to the septum **(C),** but still defined as an anterior territory MI has associated right bundle-branch block rather than QWs. RV = right ventricle.

The relationship between QW/NQW MI and a simple transmural/non-transmural dichotomy is similar to a previous autopsy study (14), but the strength of the association between QW/NQW MI classification and the underlying size of MI is novel and not previously studied. To date, our understanding of the anatomic basis of pathologic ECG changes is based on animal work and autopsy study. In these studies, the QW/NQW MI distinction was debated at length but the use of a transmural/non-transmural dichotomy to describe MI was left unquestioned. Few studies quantified total MI size (33), but no study has correlated size with the presence of QW. Using autopsy to quantify MI has a number of limitations because the study population is highly selected and unrepresentative; patients after sustained periods of cardiogenic shock with global subendocardial MI or acute second events are common, and there is often no recent ECG. The rate of previous silent infarction may be high even when only first myocardial infarcts are studied—in one study of acute MI, fatal transmural MIs had a 15% previous silent MI rate, and non-transmural MIs 51% (14). Analysis typically consists of macroscopic inspection, which may miss the changes of acute MI and of a limited number of slices only.

Late gadolinium enhancement CMR has a number of advantages over autopsy study for the investigation of MI and its relationship to the ECG. It is non-invasive with global myocardial coverage. It can detect MI down to $<1\%$ of the total myocardial mass (34), correctly identify subendocardial MI unlike lower resolution nuclear and echo techniques (35), and also detects previous silent MI. These characteristics suggest that late gadolinium enhancement CMR may allow a reinterpretation of the anatomic and pathologic basis of the ECG and may result in new, more powerful ECG criteria for clinical and research application. There has been a previous study of late gadolinium enhancement CMR in identifying MI by Wu et al. (19). This study showed in 82 patients with a range of sizes and types of MI, that late gadolinium CMR could detect 91% of MIs at 3 months (13 NQW) and 100% of MIs at 14 months (8 NQW). Only 12 of 29 patients with QW had transmural MI. However, there was no analysis relating size of MI to the presence of QW, nor a breakdown into coronary territories. Finally, although it is well established that infarct size is correlated with outcome (36), these studies have not shown whether the transmural extent of MI is independently predictive of outcome compared with MI size.

CONCLUSIONS

It is the total size rather than the transmural extent of MI that produces the ECG changes of QW MI. The division of MI into QW or NQW is useful because the presence of QWs predicts a lower ejection fraction and a larger MI. The division of MIs into transmural or non-transmural is less meaningful because MIs are rarely simply one or the other.

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